WHAT IS CLAIMED IS:

1. A method for treating or inhibiting the development of a disease, disorder or condition, which is associated with increased capillary permeability and white blood cell extravasations and selected from brain inflammation and sepsis, or symptoms thereof, comprising administering to a mammal in need thereof a therapeutically effective amount of a leukotriene C4 and D4 receptor antagonist or a pharmaceutically acceptable salt thereof, which does not cross or minimally crosses the blood brain barrier to treat or inhibit the development of the disease, disorder or condition, or symptoms thereof, with the proviso that when the method is treating or inhibiting brain inflammation, the brain inflammation is not caused by focal cerebral ischemia.

2. The method of claim 1, wherein the disease, disorder or condition, or symptoms thereof, treated or inhibited is brain inflammation, wherein the brain inflammation is not caused by focal cerebral ischemia.

3. The method of claim 2, wherein the brain inflammation is brain edema.

4. The method of claim 2, wherein the brain inflammation is a stroke.

5. The method of claim 2, wherein the brain inflammation is due to trauma to the brain.

6. The method of claim 5, wherein the trauma to the brain results from surgery.

7. The method of claim 2, wherein the brain inflammation is caused by an infection.

8. The method of claim 2, further comprising repeating the administering step until the white blood cell count reaches a normal level in cerebrospinal fluid.

9. The method of claim 1, wherein the administering step is performed pre-operatively before brain surgery or before an invasive brain operation.

10. The method of claim 1, wherein the leukotriene C4 and D4 receptor antagonist is pranlukast.

11. The method of claim 1, wherein the therapeutically effective amount of pranlukast is in a range of about 100 mg/day to 2000 mg/day.

12. The method of claim 1, wherein the therapeutically effective amount of pranlukast is in a range of about 200 mg/day to 1000 mg/day.

13. The method of claim 1, wherein the therapeutically effective amount of pranlukast is in a range of about 400 mg/day to 800 mg/day.

14. The method of claim 1, wherein the mammal is a human.

15. The method of claim 1, wherein the disease, disorder or condition treated or inhibited is sepsis or symptoms thereof.

16. The method of claim 15, wherein sepsis is due to Severe Acute Respiratory Syndrome (SARS), West Nile Fever, bacterial food poisoning, influenza encephalitis, cerebral meningitis, or arachnoiditis.

17. A method for treating or inhibiting the development of sepsis or the symptoms thereof, comprising administering to a mammal in need thereof a therapeutically effective amount of a leukotriene C4 or D4 receptor antagonist, or a pharmaceutically acceptable salt thereof.

18. The method of claim 17, wherein the mammal is a human.

19. The method of claim 17, wherein the leukotriene C4 and D4 receptor antagonist is pranlukast.

20. The method of claim 17, wherein the sepsis is selected from the group consisting of Severe Acute Respiratory Syndrome (SARS), West Nile Fever, bacterial food poisoning, influenza encephalitis, cerebral meningitis, and arachnoiditis.

21. A method for treating or inhibiting the development of brain inflammation, or the symptoms thereof, comprising administering to a mammal in need thereof a therapeutically effective amount of a leukotriene C4 and D4

receptor antagonist or a pharmaceutically acceptable salt thereof, which does not cross or minimally crosses the blood brain barrier to treat or inhibit the development of brain inflammation or symptoms thereof, wherein when the leukotriene C4 or D4 receptor antagonist is pranlukast or a pharmaceutically acceptable salt thereof, the therapeutically effective amount of pranlukast is in the range of about 400 mg/day to 800 mg/day.

22. A method for screening an inhibitor of increased capillary permeability, comprising:

administering a potential candidate inhibitor compound to a non-human mammal before or after an inflammation-inducing agent is introduced into the subarachnoid space via a cannula inserted therein through the dura mater of the brain of the non-human mammal;

measuring the amount of cerebrospinal fluid collected through the cannula; and

determining from the measured amount of collected cerebrospinal fluid if the potential candidate inhibitor compound is an inhibitor of increased capillary permeability.

23. The method of claim 22, wherein the inflammation-inducing agent is selected from the group consisting of arachidonic acid, prostaglandin, thromboxane, histamine, LPS, dextran, bradykinin, carrageenan, leukotriene, TNFα, IL-1β, and IL-6.

24. The method of claim 22, further comprising measuring the white blood cell count in the cerebrospinal fluid collected.